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REFER TO:

Tbc. Research Laboratory,
411 E. 69th St., New York 21, N. Y.

March 9, 1949.

Dr. Joshua Lederberg,
Department of Genetics,
The University of Wisconsin,
College of Agriculture,
Madison 6, Wisconsin.

Dear Josh:

Thanks an awful lot for sending the stock of mutants; you certainly do have a talent for translating thoughts into action! It is very difficult to interrupt the chain of experiments which flow so naturally from one to the next, but Werner or I will definitely be starting on K-12 very shortly.

Some spurious excitement filled the lab this past week. We just got a mutant which responds to alpha-amino-butyric acid, and also grows very well on casein hydrolysate. I had visions of identifying a new amino acid in animal protein but the spot tests, which at one day showed growth only on this compound, showed response to isoleucine on further incubation. This still is an interesting problem because the bug requires about three times as much isoleucine as it does alpha-amino-butyric, and it is very hard to explain these findings in terms of possible impurities. I am going to try to track this down a little farther but certainly don't have any evidence with several protein hydrolysates that cannot be accounted for at present by their contents of isoleucine.

I have been continuing with the phenomic barrier to back mutation, switching to a more quantitative type of experiment on pour plates, and have obtained even more dramatic results with the tryptophane-requiring mutant than with the pantothenic one that I showed in Chicago. Pantothenic seems to be a poor one to work with since in some experiments it grows a bit in minimal medium, and in others not. I presume the amount of available stored material varies with the physiological state. The tryptophane mutant, however, seems to be absolute and well suited to this kind of analysis.

My best regards to Esther,

Sincerely,

Bernard D. Davis

BDD/hl

P.S. - Incidentally, the back mutation of the tryptophane mutant was not promoted, either in streaks or on pour plates, by the presence of a purine requiring mutant growing on a limited amount of adenine. ~~This~~ was promoted only by tryptophane itself. This strengthens my confidence in the original interpretation. I don't think this necessarily should discourage one from attempting quantitative frequency studies with reverse mutations. The number of UV induced mutations to tryptophane independence so far exceeds the spontaneous ones that one should not have to worry about the correction for plate mutants that might be produced by adding a little tryptophane to the medium. I do wonder, however, how much validity there would be to measurement of back mutation rate on minimal agar. I note that Guthrie has been carrying out such studies.

B.D.D.